WHAT CAUSES APPENDICITIS?*

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THE subject of appendicitis has been so often discussed that it requires a great deal of courage on my part to bring it before this society for further consideration. All of you have seen many cases of appendicitis, have formed your own conclusions of the nature and treatment, and each of you is so convinced that his opinion is the correct one, that I am simply putting my hands in a hornets' nest when I attempt to modify these ideas.

My position in presenting a new explanation, based entirely on comparative studies in pathology, is made all the more difficult by the lack of clinical material, hospital- and laboratory-facilities. But we often make a diagnosis where the chief symptoms are absent, guided only by our medical intuition and where the clinical observation proves the correctness of our judgment.

Before considering this matter I would like to state that the term appendicitis is a misnomer. Appendicitis means inflammation of the appendix; but the anatomists know only the *appendicæ epiploicæ* and a *processus vermiformis*. Therefore an inflammation in this organ should be termed epi-para or perityphlitis. I will employ the term appendicitis, however, the long use of which has established itself *ex abuso,* because this word brings immediately before your mind's eye the entire picture of this disease in its manifold forms and variations—you know them all.

But what causes appendicitis? Many have not thought at all about this subject; some have investigated the matter and formulated theories even as grotesque as they are contradictory. When we find many theories, we may be sure that none are correct, just as many remedies for one disease is proof of the inefficiency of them all. If one theory is correct, why formulate new ones?

The old theory, endorsed by such an eminent man as Billroth, was that appendicitis is caused by foreign bodies. Cherrystones, fishbones, etc., were searched for, and when, instead of cherrystones, etc., fecal concretions were found, the latter were considered the cause of the disease. In the meantime, the operation for appendicitis became more frequent and many of the extirpated processi vermiformes contained neither cherrystones nor fecal concretions. What caused the inflammation in these cases was the universal question. Others stated that small microscopical particles of agateware or oystershells found their way into the processus vermiformis and produced the disease. In other instances ascaris lumbricoides, oxyuris vermicularis and trichocephalus dispar were found.

How do fecal concretions develop in the processus vermiformis? I believe that every peristaltic impairment is capable of producing fecal concretions. The feces enter the processus vermiformis just as readily as other parts of the intestines, but escape with more difficulty because the peristaltic power is here weaker and the canal has no distal opening. It is only necessary that the peristalsis be impaired or entirely absent for a few days, as occurs in cases of peritoneal irritation, for the feces to remain dormant in the processus vermiformis. They become harder and the re-established peristalsis can only with much difficulty expel them. Small particles, however, remain, become harder and harder, and a fecal calculus is formed. In a case of extrauterine pregnancy I had the opportunity to observe the formation of such a concretion. The patient showed, following an internal hemorrhage, signs of peritonitis, suppression of stool and gas. These symptoms disappeared in a few days and 12 days later the patient consented to an operation. I found that the processus vermiformis was partially filled with feces; the cecal end contained soft matter, which was segmented by the peristalsis, while in the distal end the fecal contents were hard. Such a concretion could only be removed by operative intervention. The development of fecal concretions is therefore very simple and comparatively frequent. That fecal concretions are not the real cause of appendicitis is proven by the fact that we find them present in only about 20 per cent. of the patients operated upon.

It is not my purpose to here enumerate all the theories; suffice it to say that many claim to have found the key in the histology of the processus vermiformis, others in its anatomy. The artery which supplies the processus vermiformis was by some held responsible for the mischief. Others claimed it depended upon the length of the processus vermiformis. Some assert that its various positions is the sole factor causing disease, others swear it is nothing but a retrograde metamorphosis. Again some few see in the pressure of the kidney upon the processus vermiformis the explanation, and so the theories pile up upon each other *ad infinitum*. My attempt to collect the literature published upon this subject encountered such voluminous and contradictory material that I gave up in despair. Is any one of the numerous theories correct? I think not, although each of these conditions above quoted may have some slight influence in producing the disease. In my opinion, there is but one cause for appendicitis; namely, *loss of tissue and subsequent infection.* The fecal concretion is not the cause of appendicitis, but produces a lesion of the tissue, which becomes subsequently infected. If no infection ensues, then the fecal concretion or foreign body causes no further trouble. We find the same in gallstones. Gallstones may be

* Read before the San Francisco County Medical Society.

carried during a lifetime without any inconvenience. Indeed, they are so common that we find them in 10 per cent. of autopsies. Only when infection occurs do they become a source of disease. How can the length of the processus vermiformis be held responsible for its inflammation? Or its blood supply, its histology or its anatomy? According to what law? Do we find any analogy in pathology for this?

Nowhere in surgery does one find infection occurring without a primary lesion. No surgeon of today accepts the old ideas about idiopathic erysipelas or tetanus, because he knows that a point of entrance, be it even so small that it escapes discovery, is necessary to make infection possible. Normal skin and normal mucous membranes are not penetrable by infectious germs. Only in appendicitis do we attempt to make exception to this law. Why? How does the lesion in the tissue occur? In many ways. It struck me that more boys than girls, more men than women were afflicted with appendicitis, which disease is more prevalent in America than in Europe. These facts started me to thinking, especially as appendicitis in women should be relatively more frequent, owing to the neighboring adnexa, so frequently diseased. This is not the case, however. Further, I was impressed that nurses in a certain hospital, who lifted the patients upon the operating-table, were frequently attacked with and operated upon for appendicitis. Upon further investigation, I found similar conditions in another hospital. It is a fact that appendicitis is frequent among students of Stanford University. Cases of appendicitis following traumatisms, as a blow, lifting a heavy weight suddenly, etc., are not rare. In the above mentioned cases the loss of tissue was produced by capillary hemorrhages through overexertion and the subsequent infection developed the appendicitis. By means of this assumption we can explain why more boys than girls, more men than women, more here than in Europe suffer from appendicitis. Boys expose themselves more to physical overexertion than girls. The same applies to men, and the fact that physical sports are more cultivated here than in Europe accounts for the greater frequency of appendicitis in America. The fact that capillary hemorrhages may occur after physical overexertion is well known to oculists. Rupture of the conjunctival capillaries follows such efforts as severe coughing, sneezing, straining by stool or lifting heavy weights quite commonly. Such a capillary hemorrhage may likewise occur in the processus vermiformis through sudden increase of the intraabdominal pressure, and indeed here more readily when such predisposing conditions as abnormal position, shape, blood supply and histological structure are present. It is, however, not necessary to assume that these small hemhorrages occur more frequently in the processus vermiformis. They may as well occur in other parts of the intestinal tract, but here no further complications ensue. And this is readily explained. The intestinal tract is an open canal, while the processus vermiformis is a blind sac. In the former the normal secretion of mucus is considerable and the contents are kept in constant movement, while in the latter peristalsis is slow and the liability to infection is considerably greater. We may compare the conditions existing in the processus vermiformis with those in a fistula. A fistula seldom heals unless converted into an open wound, because the secretions collect, producing constant irritation. All these conditions favor infection of the processus vermiformis. The severity of the disease will depend upon the nature of the infection.

The foregoing theory readily explains why infectious diseases such as tonsilitis may sometimes be followed by appendicitis. Pathology teaches us that internal capillary hemorrhages in different organs occur in the various infectious diseases. Thus in scarlet fever we find hemorrhages in the kidneys and intestines, in acute articular rheumatism hemorrhages in the mediastinum, peri- and endocardium, the pleura, the spleen and the serosa of the intestine are frequent; also, in influenza effusions of blood occur in different organs. These hemorrhages result from the action of the toxins and can be secondarily infected by the same germ which caused the original disease, as, for example, streptococci, or more commonly, the bacterium coli. There remains now but to explain the appendicitis due to foreign bodies. This is also quite simple. If the foreign body is sharp, the mucosa is easily wounded and the ever-present coli bacillus will cause the infection. If the foreign body is dull, it may cause a pressure necrosis, thus making infection possible. The development of chronic appendicitis with acute exacerbations finds also its natural explanation. The primary lesion is infected with germs of moderate virulence. In the ensuing battle nature obtains the upperhand and the acute attack subsides. The lesion, however, does not heal, but is transformed into a small, granulating ulcer (I beg you to remember that the conditions in the processus vermiformis are similar to those in a fistula). Any new irritation of this wound will facilitate another infection and therefore a fresh attack. Should, however, this little ulcer heal entirely, then a scar forms with subsequent stenosis. If infection takes place through highly virulent germs, then the whole processus vermiformis is converted into a phlegmon. Summing up, *appendicitis is caused through loss of tissue with subsequent infection. This loss of tissue can be caused by capillary hemorrhages due to infectious diseases or overexertion, or by foreign bodies.* No appendicitis without infection, no infection without loss of tissue.